



Anaesthesia for Patent Ductus Arteriosus (PDA) ligation

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Introduction

Many hospitals now have surgeons who are willing to perform complex procedures formerly undertaken in only a few specialist institutions. Not all of these hospitals have anaesthesiologist with training in paediatric cardiovascular anaesthesia.

In order to provide safe anaesthesia for children with congenital heart disease, the anaesthesiologist must have a detailed understanding of the pathophysiology of the lesion and the pharmacology of drugs being used.

The Ductus Arteriosus (PDA) is a persistent foetal communication from the main pulmonary artery (PA) to the descending aorta. The incidence of this defect is 1 in 2500 live full-term births, accounting for approximately 10% of all congenital heart defects. 1, 2, 3

Anatomy

The isolated PDA arises from the anterior surface of the main pulmonary artery near its junction with the left pulmonary artery and joins the posterior descending aorta after the origin of the left subclavian artery. The shunt between the aorta and the PA can be restrictive or non restrictive. The magnitude and direction of flow are determined by the pressure in the aorta and resistance to flow in the two vascular beds, pulmonary and systemic. With non-restrictive PDA, the normal low pulmonary vascular resistance leads to an increased pulmonary blood flow and rise in pulmonary vascular pressures while systemic blood flow is frequently reduced. 1, 2

Pathophysiology

At birth, several important changes occur in the heart and peripheral circulation. The circulation of the newborn infant is in transition from the foetal to the adult pattern. It can be shift back to the foetal pattern by the presence of congenital heart disease, prematurity, anaesthesia, hypoxia and other

conditions. Shunting through the PDA and across the foramen ovale is characteristic of foetal circulation.

After birth and the cessation of placental circulation, an increase in systemic vascular resistance occurs. Pulmonary vascular resistance decreases 75 % and pulmonary blood flow increase 450 % with the beginning of neonatal respiration. From a prenatal pressure of 70/45, the pulmonary artery pressure decrease to 50/30 at 24 hours after birth and to 30/ 12 a few days later. Vasoactive drugs, including bradykinin, prostaglandins and endothelium derived relaxing factor contribute to pulmonary vasodilation. Oxygen and ventilation contributes to increases in the release of these substances.

The PDA begins to close within 10-15 hours of birth as a result of the increased arterial oxygen pressure and vasoactive factors. The ductus is physiologically closed by the second day of life in normal infants, but we must remember that it can reopen in response to hypoxia. In neonates, acute moderate hypoxia increases cardiac output if metabolic acidosis is absent. However, in the presence of metabolic acidosis, cardiac output decreases. Acute severe hypoxia causes bradycardia. Other changes associated with hypoxia are decreased myocardial contractility, increased pulmonary vascular resistance and reopening of the ductus with shunting. Permanent anatomic closure of the ductus normally occurs within the first 3 weeks of life. The haemodynamics of PDA are similar to those of Ventricular Septal Defect in that the amount of left to right shunt is governed by size of the defect and vascular resistance. When the ductus is large, the magnitude of the shunt is determined by the ratio of pulmonary vascular resistance to systemic vascular resistance. 1, 2

Natural History and Clinical Manifestations

The natural history of PDA is similar to that of other lesions with a left to right shunt. In most patients, congestive heart failure, manifested by tachypnoea, fluid retention, hepatomegaly and cardiomegaly are present. An increase in pulmonary vascularity may be evident on chest X-rays. There is always a risk of bacterial endocarditis and this is a primary indication for closure even of small asymptomatic PDA. The risk persists for at least 3 years after the closure. 1 Children with PDA have a continuous, machinery-type murmur in the left first intercostals space. Due to the left ventricular failure, fatigue and dyspnoea most children with a PDA are malnourished. Because of the low resistance shunt through the pulmonary vasculature, the systemic arterial pulse pressure is often wide, giving the impression of bounding peripheral pulses.

Preoperative evaluation

During pre-operative assessment it is necessary to look carefully for evidence of problems such as respiratory infections and congestive heart failure. Anesthetising a child with a respiratory infection may lead to a higher incidence of complications if intubation is required or the airway is manipulated. Laryngospasm or bronchospasm may occur as a response to glottic or subglottic mucosal stimulation. 4 Upper respiratory tract infections cause a striking increase in bronchial reactivity that appears to persist for 3 to 4 weeks after infection. 5

As a minimum, blood gas measurements, haematocrit, blood chemical and glucose analysis should be performed to ensure that the infant is adequately prepared for surgery.

Anaesthetic management Non surgical closure of the PDA is possible in selected patients. Administration of indomethacin will produce functional closure of the Ductus in 50-70 % of infants. 1, 3

Catheter closure of PDA has been performed since 1977 in developed countries and, since the 1990s, it has also been a common procedure in our country limited only by economic considerations (the cost and availability of the devices). This non-surgical closure of the defect eliminates the need for a thoracotomy and prolonged hospital stay and can be performed under sedation in a catheter laboratory.

The anaesthetic technique for surgical repair is variable but in severely ill patients the administration of fentanyl and a non-depolarizing muscle relaxants provides adequate analgesia and surgical operating conditions. Ventilation is maintained with a mixture of air and oxygen. This combination is well tolerated even by critically ill infants with congestive heart failure.

We use on induction dose of fentanyl of 5-10 micrograms/kg and 30-50 micrograms/kg in total for maintenance. In haemodynamically stable children, this technique usually allows extubation at the end of procedure. Ketamine, isoflurane and midazolam are useful drugs that can be use to supplement the anaesthetic. 1, 2, 3, 6, 7

Most patients have undergone fluid restriction and diuretic treatment whilst being prepared for surgery and may require volume replacement before induction. We use Ringer lactate 10 ml/kg for this purpose. Per-operative hydration is accomplished with Ringer lactate at 5 ml/kg/hour.

The surgical approach to the PDA is usually through a left postero-lateral thoracotomy. Intercostal nerve blocks at the end of the procedure are effective in controlling post-operative pain and can be done by the surgeon under direct vision before the chest is closed.

Ductus ligation is usually accompanied by an increase in diastolic pressure and variable and transient effects on systemic pressure. Surgical complications are rare but include damage to the recurrent laryngeal nerve and inadvertent ligation of either the aorta or the left pulmonary artery, both of which may be similar in size to the ductus in the neonate. 2 Postoperatively the child requires a chest tube for 24 hours and may stay in the hospital for 3-5 days.

Conclusions

The peri-operative course of each patient is unique; however, certain problems and complications are often associated with each specific congenital cardiac condition and should be anticipated in order to ensure optimal anaesthesia management.

References

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